

Dyslipidemia in hemodialysed patients of chronic renal failure

Fating Prasanna M^{1*}, Tadas Arun K², Katore Sarika D³, Tadas Swati A⁴

{¹Assistant Professor, ³Jr. Medical Officer, Department of Biochemistry} {⁴Assistant Professor, Department of Physiology}
Government Medical College, Nagpur, Maharashtra, INDIA.

²Professor and HOD, Department of Biochemistry, Shri Vasantrao Naik Government Medical College, Yawatmal, Maharashtra, INDIA.

Email: drprasannafating@gmail.com

Abstract

Background: Cardiovascular disease (CVD) is major cause of mortality and morbidity among patients with Chronic kidney disease (CKD). More than 50% of patients with CKD die due to cardiovascular complication and dyslipidemia is an independent risk factor for CKD. The incidence of coronary artery disease is seen in 28 percent of dialysis patients. So we considered to study this relation of chronic renal failure (CRF) and lipid profile in dialysed patients. **Aim:** To estimate lipid profile in hemodialysed patients of chronic renal failure. **Settings and Designs:** This cross sectional study was undertaken in the Department of Biochemistry and kidney Unit, Department of Medicine and Department of Nephrology of Government Medical College and superspeciality hospital Nagpur, Maharashtra (India). **Material and Methods:** Total cholesterol (TC), Triglycerides (TG), High density lipoprotein (HDL), low density lipoprotein (LDL), very low density lipoprotein (VLDL), atherogenic ratio i.e. TC/HDL, LDL/HDL, was assessed in hemodialysed CRF patients (n=50) and healthy individuals (n=50). Mean of biochemical parameter were compared by performing student's t-test. **Results:** Values of triglyceride, LDL-C and TC/HDL, LDL/HDL were significantly higher in hemodialysed patients of CRF but values of HDL-C (p<0.001) were significantly lower in hemodialysed patients of CRF as compared to controls. **Conclusion:** Atherogenic dyslipidemia was more pronounced in hemodialysed patients as compared to normal patients. **Keywords:** Cardiovascular disease, Dyslipidemia, cardiovascular disease, CKD, Atherogenic ratio., CRF.

*Address for Correspondence:

Dr. Fating Prasanna M, Assistant Professor, Department of Biochemistry, Government Medical College, Nagpur, Maharashtra, INDIA.

Email: drprasannafating@gmail.com

Received Date: 11/10/2014 Accepted Date: 21/10/2014

Access this article online

Quick Response Code:



Website:

www.statperson.com

DOI: 02 November
2014

INTRODUCTION

Chronic renal failure (CRF) also known as chronic kidney disease (CKD) is a progressive loss in renal function over a period of months or years. Chronic kidney disease is affecting millions of people worldwide¹. It has been estimated that prevalence of CKD in India is 785 people per million population². The altered functioning of the renal system affects every organ of the body. The severity of the consequences of CRF has however undergone

profound changes since the introduction of dialysis. Cardiovascular disease (CVD) is major cause of mortality and morbidity among patients with CRF. More than 50% of patients with CRF die due to cardiovascular complication and dyslipidemia is an independent risk factor for CRF in undialysed as well as dialysed patients³. In this study we studied the dyslipidemia in hemodialysed patients of chronic renal failure.

MATERIAL AND METHODS

Subjects

The present study has been carried out in Govt. medical college and hospital, Nagpur, India from January 2012 to July 2013. The study protocol was approved by the Institutional Ethical Committee. Informed written consent was obtained from all the study subjects enrolled in the study. Study sample was consisted of total 100 individuals; 50 diagnosed patients of hemodialysed CRF in the age group of 30-70 years of either sex, admitted in kidney unit of medicine department in the institute and Nephrology department in Govt medical college and

superspeciality hospital, Nagpur and 50 age and sex matched controls were also selected for study.

Inclusion Criteria

- Diagnosed patients with CRF
- Age >30 yrs and <70yrs
- Patients on hemodialysis for more than 3 months

Exclusion Criteria

- Age < 30 yrs and >70yrs
- Patients on peritoneal dialysis
- Nephrotic syndrome
- Ischemic heart disease
- Hepatic diseases
- Hypothyroidism
- Familial hypercholesterolemia
- Patients on hyperlipidemic drugs
- Those who did not consent to participate in the study.

Sample Collection

Five ml of venous blood samples were collected from the subjects in plain bottles after an overnight fast of 12

hours. The samples were allowed to stand for half an hour. The serum was separated and serum lipid profile was estimated on the same day.

Laboratory Analysis

Estimation of serum cholesterol was done with the kit based on cholesterol oxidase peroxidase (End Point) method⁴. Triglyceride estimation was done with the kit based on Glycerol 3 phosphate oxidase peroxidase (End Point) method⁵. HDL-C estimation was done with the kit based on precipitation method⁶. The estimation was done on TRANSASIA ERBA CHEM□5 Plus Semi□Automatic Analyzer. LDL-C was calculated by Friedewald's formula⁷.

Statistical Analysis

Statistical data was recorded on Microsoft Excel programme. Data was analysed by using statistical software STATA version 10.0 All continuous variables were presented as mean \pm standard deviation. Lipid parameters in two groups were compared by performing student t-test.

Table 1: Lipid profile in hemodialysed patients of CRF and Controls

Variable	Control Group I (n=50) (Mean \pm SD)	Hemodialysed Group II (n=50) (Mean \pm SD)	P Value
Triglycerides (mg%)	134.4 \pm 22.70	242.4 \pm 24.57***	<0.001
Total Cholestrol (mg%)	176.4 \pm 22.42	183.2 \pm 14.29	>0.05
HDL-C (mg%)	42.56 \pm 9.01	27.62 \pm 3.20***	<0.001
LDL-C (mg%)	106.9 \pm 22.79	107.1 \pm 13.90	>0.05
VLDL-C (mg%)	26.89 \pm 4.54	48.48 \pm 4.91	<0.001

*=(p<0.05); **=(p<0.01); ***=(p<0.001). n=Number of subjects; SD= Standard deviation

RESULTS AND ANALYSIS

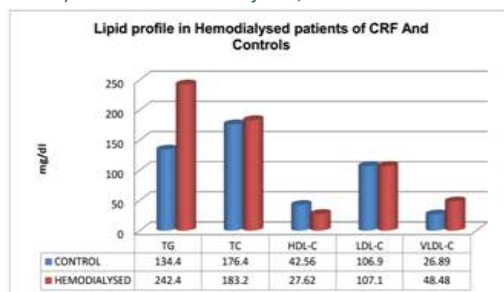
The table (I) shows serum lipid profile in controls and hemodialysed patients of CRF. There was higher serum triglyceride observed in hemodialysed patients of CRF as compared to controls (p<0.001). There was low serum HDL-C observed in patients of hemodialysed CRF as compared to controls (p<0.001). There was higher serum VLDL-C observed in patients of hemodialysed CRF as compared to controls and the difference was statistically highly significant (p<0.001). There was higher serum

total cholesterol observed in hemodialysed CRF patients as compared to controls but the difference was statistically non significant (p>0.05). There was higher serum LDL-C observed in patients of hemodialysed CRF as compared to controls but the difference was statistically non significant (p>0.05). Table II shows the atherogenic ratio. The serum LDL-C/HDL-C and serum TC/HDL-C were higher observed in patients of hemodialysed CRF as compared to controls (p<0.001).

Table 2: Atherogenic ration in hemodialysed patients of CRF and controls

Variable	Control Group I (n=50) (Mean \pm SD)	Hemodialysed Group II (n=50) (Mean \pm SD)	P Value
LDL-C/HDL-C	2.65 \pm 0.91	3.92 \pm 0.67***	<0.001
TC/HDL-C	4.31 \pm 1.05	6.70 \pm 0.84***	<0.001

*= (p<0.05); **= (p<0.01); ***=(p<0.001). n=Number of subjects; SD= Standard deviation



DISCUSSION

It was suggested that, the most important pathophysiological mechanism underlying the development of hypertriglyceridemia in CKD was accumulation of triglyceride rich lipoproteins i.e. VLDL, IDL, chylomicrons and their remnants, either due to increased synthesis (promoted by insulin resistance) or decreased catabolism. But the decreased catabolism was found to be main mechanism. It was also supported by Lacquaniti A *et al*⁸, Tsimihodimos V *et al*⁹ and Wanner C *et al*¹⁰. Several authors have suggested that the primary metabolic defect appears to be a defective catabolism of triglyceride rich lipoproteins (primarily VLDL) by the enzymes lipoprotein lipase and hepatic lipase^{11,12}. In previous studies it was found that in CRF, there were metabolic abnormalities of plasma lipoproteins. This alteration in protein structure of lipoprotein is due to protein carbonylation and accumulation of advanced glycation end products in the patients of CRF^{13,14}. Lower HDL mean value is primarily due to CRF induced dysregulation of several important proteins i.e. LCAT, CETP, ACAT, and apo A I and apo AII¹⁵⁻²¹. The probable mechanism of hypertriglyceridemia in hemodialysed patients is due to the repeated use of heparin as an anticoagulant affecting lipoprotein metabolism. Heparin releases LPL from the endothelial surface and thus its chronic use may result in LPL depletion and defective catabolism of triglyceride rich lipoproteins²². Hemodialysed patients exhibit a more atherogenic lipid profile, such as increased serum TG and decreased apolipoprotein (apo) A and/or HDL-C than healthy controls without chronic kidney disease (CKD). These findings were supported by Solski J *et al* (2000)²³, Jeong TK *et al* (1998)²⁴, Siamopoulos KC *et al* (1995)²⁵ and Steele J *et al* (1989)²⁶.

CONCLUSION

From our study, it appears that there is a derangement of lipid profile in hemodialysed CRF patients. We propose that regular estimation, at least twice yearly; of lipid profile in these patients may help to correct these parameters at earliest and will save patients from the disastrous effects like cardiovascular diseases and stroke.

REFERENCES

1. Angelantonio ED, Danesh J, Eiriksdottir G, Gudnason V. Renal Function and Risk of Coronary Heart Disease in General Populations: New Prospective Study and Systematic Review PLoS Medicine September 2007; 4(9):1497-1507 www.plosmedicine.org
2. Sanjay Kumar Agarwal, Suresh Chand Dash, Mohammad Irshad, Sreebhuaan Raju, Ravinder Singh and Ravinder

- Mohan Pandey Prevalence of chronic renal failure in adults in Delhi, India. Nephrol Dial Transplant (2005) 20: 1638-1642
3. Kaya Y, Ari E, Demir H, Soylemez N, Cebi A, Alp H *et al*. Accelerated atherosclerosis in haemodialysis patients; correlation of endothelial function with oxidative DNA damage, Nephrol Dial Transplant (2012) 27: 1164-1169.
4. Cholesterol reagent set [Kit insert]. Thane (India): Accurex Biomedical Pvt. Ltd; 2009
5. Triglyceride reagent set [Kit insert]. Thane (India): Accurex Biomedical Pvt. Ltd; 2009.
6. HDL-cholesterol reagent set [Kit insert]. Thane (India): Accurex Biomedical Pvt. Ltd; 2009.
7. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972; 18:499-502.
8. Lacquaniti A, Bolignano D, Donato V, Bono C, Fazio MR, Buemi M. Alterations of Lipid Metabolism in Chronic Nephropathies: Mechanisms, Diagnosis and Treatment. Kidney Blood Press Res 2010; 33:100-10.
9. Tsimihodimos V, Dounousi E, Siamopoulos KC. Dyslipidemia in Chronic Kidney Disease: An approach to pathogenesis and treatment. Am J Nephrol 2008; 28:958-73.
10. Wanner C. Importance of hyperlipidaemia and therapy in renal patients. Nephrol Dial Transplant 2000; 15(5):92-6.
11. Blaton V. Dyslipidemia at chronic renal failure. eJIFCC 2009; 20(1):58-68.
12. Chan DT, Dogra GK, Irish AB. Chronic kidney disease delays VLDL apoB-100 particle catabolism: potential role of apoC-III. J Lipid Res 2009; 50:2524-31.
13. Galli F, Benedetti S, Floridi A *et al*. Glycoxidation and inflammatory markers in patients on treatment with PMMA-based protein-leaking dialyzers. Kidney Int 2005; 67:750-759
14. Himmelfarb J, McMonagle E. Albumin is the major plasma protein target of oxidant stress in uremia. Kidney Int 2001; 60:358-363
15. Guarnieri GF, Moracchiello M, Campanacci L, Ursini F, Ferri L, Valente M *et al*. Lecithin cholesterol acyltransferase (LCAT) activity in chronic uremia. Kidney Int Suppl 1978; 8:S26-S30.
16. McLeod R, Reeve CE, Frohlich J. Plasma lipoproteins and lecithin:cholesterol acyltransferase distribution in patients on dialysis. Kidney Int 1984; 25: 683- 688
17. Shoji T, Nishizawa Y, Nishitani H, Billheimer JT, Sturley SL. Impaired metabolism of high density lipoprotein in uremic patients. Kidney Int 1992; 41:1653-1661.
18. Kimura H, Miyazaki R, Imura T, Masunaga S, Suzuki S, Gejyo F *et al*. Hepatic lipase mutation may reduce vascular disease prevalence in hemodialysis patients with high CETP levels. Kidney Int 2003; 64:1829-1837.
19. Vaziri ND, Liang K, Parks JS. Downregulation of lecithin: cholesterol acyltransferase (LCAT) in chronic renal failure. Kidney Int 2001; 59:2192- 2196.
20. Vaziri ND, Sato T, Liang K. Molecular mechanism of altered cholesterol metabolism in focal glomerulosclerosis. Kidney Int 2003; 63:1756 -1763

21. Chan CM. Hyperlipidaemia in Chronic Kidney Disease. *Ann Acad Med Singapore* 2005; 35:31-5.
22. Nasstrom B, Stegmayr B, Olivecrona G, Olivecrona T. Lipoprotein lipase in hemodialysis patients: indications that low molecular weight heparin depletes functional stores, despite low plasma levels of the enzyme. *BMC Nephrology*, 2004;5:17
23. Solski J, Kimak E, Janicka L, Ksaziek A, Janicki K.. Concentration Of Lp(A) And Other Apolipoproteins In Predialysis, Hemodialysis, Chronic Ambulatory Peritoneal Dialysis And Post-Transplant Patients, *Clin Chem Lab Med* 2000 May;38(5):421-50
24. Jeong TK, Kim HS, Nah MY, Jeong GH, Jung K, Lee SC. *Korean J Nephrol* 1998
25. Siamopoulos KC, Elisaf MS, Bairaktari HT, Pappas MB, Sferopoulos GD, Nikolakakis NG, *et al.* Lipid Parameters Including Lipoprotein (A) In Patients Undergoing Capd And Hemodialysis. *Perit Dial Int* 1995 Oct-Dec; 5(8):342-7.
26. Steele J, Billington T, Janus E, Moran J. Dept of Chemical Pathology, St Vincent's Hospital, Fitzroy, Victoria, Australia. *Atherosclerosis* 1989 Sep; 79(1):47-50.

Source of Support: None Declared
Conflict of Interest: None Declared